Letter to the Editor: Cytomegalovirus Causing Pericarditis With Tamponade in an Adolescent With Cancer

Cytomegalovirus (CMV), a common infectious agent in immunocompromised patients, rarely causes isolated pericarditis. To our knowledge, CMV pericarditis has been reported in only four patients, all adults [1–4]. Three of the four developed cardiac tamponade, and only one had a malignancy [4]. We report a case of CMV pericarditis with cardiac tamponade in an adolescent with cancer.

A 16-year-old boy with Klinefelter's syndrome was referred to St. Jude Children's Research Hospital for investigation of a large mediastinal mass diagnosed as a malignant teratoma. Metastatic workup was negative and pleural fluid contained no tumor cells. Pleural fluid cultures were negative for bacteria, fungi, and viruses at that time. The patient received carboplatin, etoposide, and ifosfamide at intervals of 21–28 days without cytokine support. He received 12 units of packed cells and 8 units of platelets, all serologically positive for CMV. Blood products were irradiated but not leukocyte-depleted.

After his fourth chemotherapy course, the patient was admitted to the hospital with fever and neutropenia. Two days later, his temperature reached 39.8°C, associated with moderate hypotension, poor capillary refill, and mental confusion. He also developed an irregular heart rate and tachycardia, and pulsus paradoxus was 14 mmHg. Electrocardiography revealed atrial flutter and marked ST and T wave changes with diffuse low voltage. A two-dimensional (2D) echocardiogram revealed a large pericardial effusion with normal indices of left ventricular function and wall thickness. Pericardiocentesis under fluoroscopic guidance yielded 240 ml of serosanguinous fluid, which was examined for cytologically and cultured for bacteria, fungi, acid fast bacilli, and viruses. A pericardial drain was inserted due to persistent tachycardia and tachypnea. The pericardiocentesis culture grew CMV on an MRC cell line after 8 days of incubation. Urine and blood were negative for the virus by shell vial assay and culture at three different times over a period of 3 weeks after the identification of CMV. The patient began receiving IV ganciclovir (5 mg/kg every 12 hours) and IV IgG (0.5 g/kg every other day). After 2 days of ganciclovir treatment, a percutaneous liver biopsy was performed due to hepatomegaly and progressive increase in serum bilirubin and transaminases, revealing nonspecific mild chronic portal triaditis without viral inclusions. Immunoperoxidase studies of biopsy tissue for CMV and hepatitis B were negative, as were viral cultures.

The patient defervesced on the third day of treatment and remained afebrile. The pericardial catheter was removed at that time without reaccumulation of fluid. Antiviral therapy was discontinued after 10 days. Subsequently, the mediastinal tumor was totally resected and chemotherapy was electively stopped. Two months later, a 2D echocardiogram showed normal left ventricular function and cardiac structures without evidence of pericardial or pleural effusion. He was on no cardiac medications, the hepatomegaly had resolved, and CBC and liver function tests were normal. He remained disease-free when last seen for follow-up 42 months after the original diagnosis.

In brief, the patient was probably exposed to CMV via multiple blood product transfusions that were not leukocyte-depleted. His potentially fatal CMV pericarditis with tamponade was managed successfully by pericardiocentesis and antiviral therapy. This case should alert pediatric oncologists, infectious disease specialists, as well as cardiologists to the existence of such viral complications in pediatric cancer patients. Such infections may become more prevalent as increasingly intensive chemotherapy is given in conjunction with cytokines and broad-spectrum antibiotics.

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